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# ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

## DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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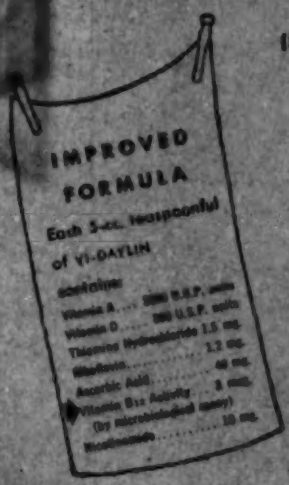


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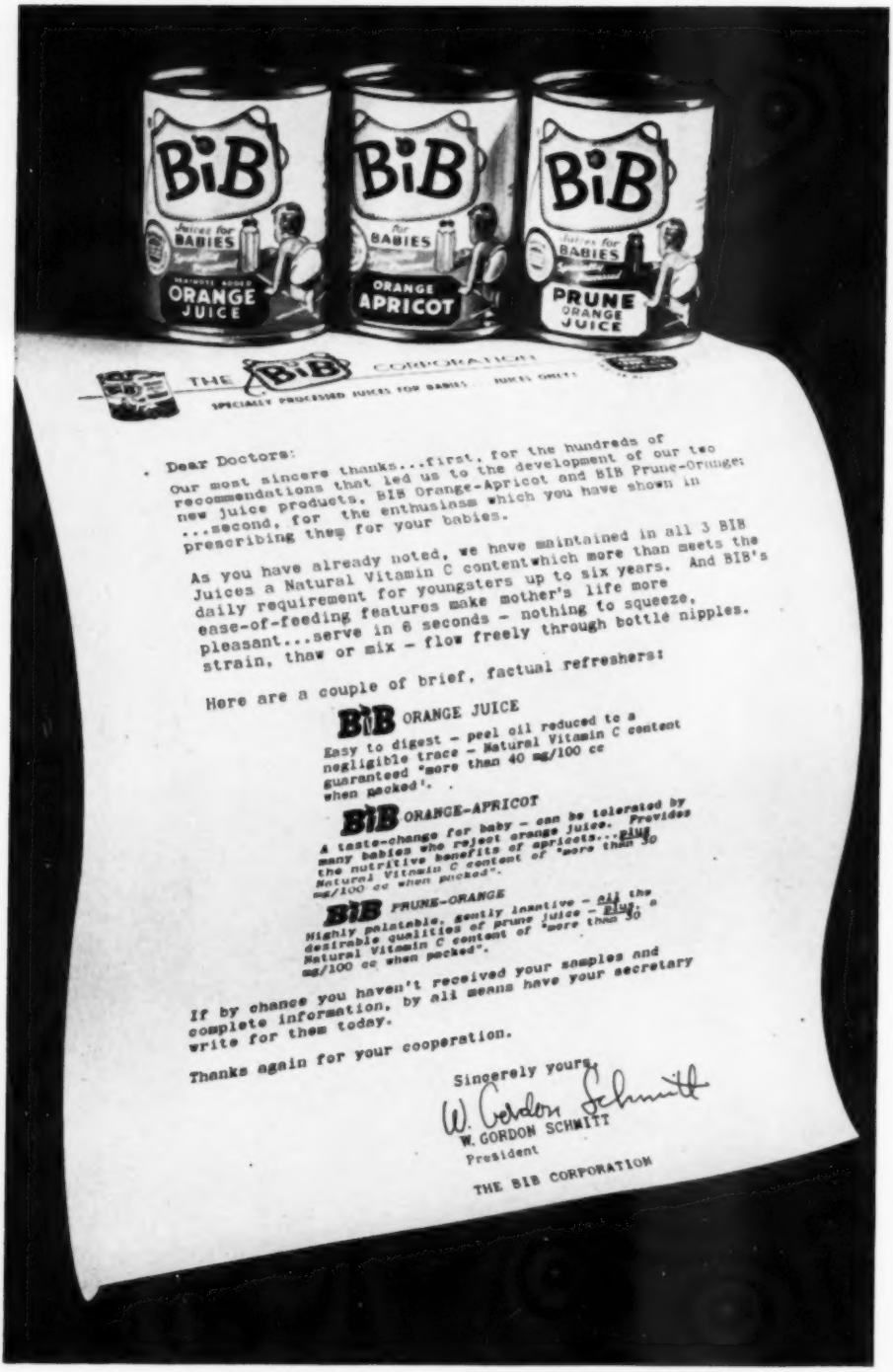
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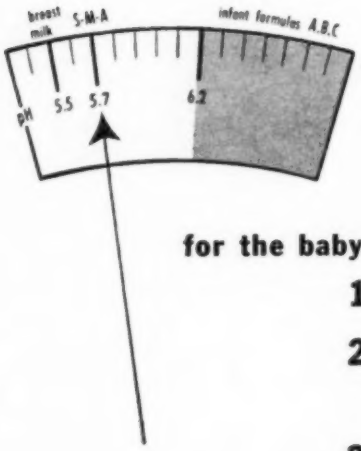


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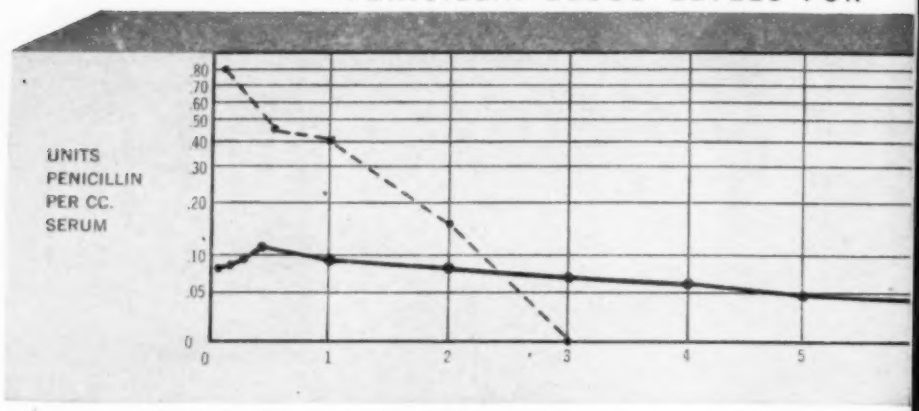


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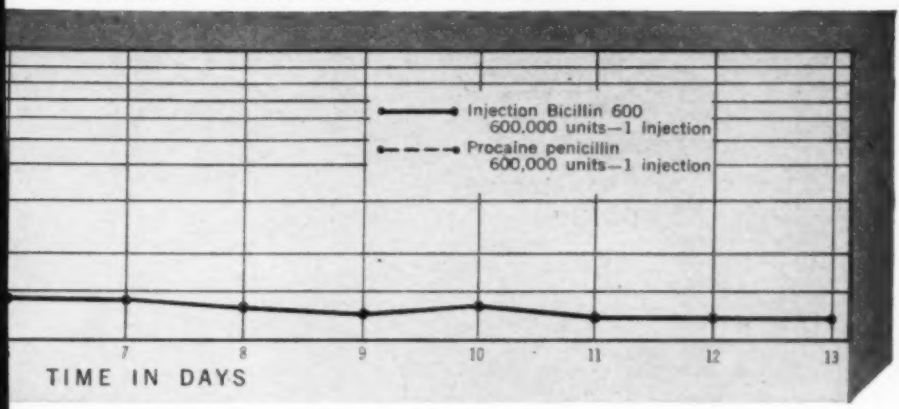


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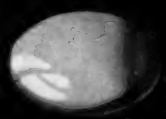

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## LIGHT MAGNESIUM CARBONATE IN THE TREATMENT OF ACUTE GLOMERULAR NEPHRITIS

### PRELIMINARY REPORT


C. L. THENEBE, M.D.

West Hartford, Conn.


One might ask, without impunity, what advantages does the new light magnesium carbonate (Merck) have over the old established magnesium sulphate treatment of acute glomerular nephritis?

Comparably, magnesium carbonate tastes much better and it is not at all nauseating. It is harmless in large doses and it might even be proven to be more effectual. Certainly, it is more desirable than the administration of the painful intramuscular injections of magnesium sulphate.

During the past two years, I have used the treatment at home upon six patients. Two other patients were treated at the hospitals. In all of the patients, there was a past history of infection. The site of focal infections was searched for and treated. Antibiotics were used when an active infection was present. When a definite anemia was existent, blood transfusions were given.

During the process of pathologically involving the kidney, the line of glomerule or tubule damage is not clearly defined. Those cases of prolonged nephrosis revealing extensive glomerular involvement at autopsy prove that there is little mark of demarcation. The cases therefore are often mixed.

In treating the acute glomerular nephritic with light magnesium carbonate, six or more liquid stools within twenty-four hours must be accomplished. In the presence of an abnormal condition, i.e.,

nephritis, it takes a much larger dose than would ordinarily be required, to accomplish this.

I start with two heaping tablespoons every four hours, to be increased or decreased, according to the results obtained. The light magnesium carbonate is mixed with chilled water.

Modified treatment is carried along at home for a period usually not exceeding five weeks. The daily number of loose stools should be at least four after the initial period of approximately eight days of more frequent stools. Wherein vomiting was not prominent, the usual house but salt-free diet was prescribed. It does follow that early treatment seems to be more helpful. Falling blood pressure and diminishing red blood cells in the urine were apparent within thirty-six hours after the start of the treatment.

## CASE REPORTS

*Case* 1. Age 8 years, female. Admitted May 7 and discharged May 16. She had an influenzal-like infection three weeks before and a unilateral suppurative otitis media two weeks before hospital admission. The ear drained for one week. Four days before admission, she developed puffiness about the eyes. The urine became increasingly bloody during this latter four-day period.

The blood pressure fell from a peak of 154 mms. systolic, the day of admission, to 120 mms. the day of discharge. There was a daily recession. The N.P.N. dropped from 35 to 30.

*Case 2.* Age 7½ years, female. Admitted May 7 and discharged May 18. Frequent attacks of tonsillitis and otitis media, 1948; adenotonsillectomy, 1949; acute ethmoiditis, January 1952.

Ten days before hospital admission, pharyngitis. Gantrisin and penicillin were prescribed. The infection subsided within three days. There was a slight exposure to the sun which produced an erythema of the facial skin five days prior to admission. Puffiness about the eyes began four days before hospital admission. During the first three days of periorbital swelling, there were *no* red blood cells in the urine (laboratory). The red blood cells appeared in the urine the day of admission. Up to the day of hospital admission, albumin 300 per 100; W.B.C. up to 50 H.P.F.; casts up to 75 H.P.F.

There was a daily fall of blood pressure from an initial average of 180 mms. to a discharge one of 112 mms.

There was only an occasional red blood cell in the urine upon the discharge day and there were some intervening days between admission and discharge wherein none were found. May 8 N.P.N. 41; cholesterol 193; serum protein 7. May 13 N.P.N. 45; cholesterol 276; serum protein 7.7. May 17 N.P.N. 39; cholesterol 217; serum protein 7.6.

The urine was consistently alkaline throughout in all cases receiving light magnesium carbonate.

SUMMARY

I hope that in the future, light magnesium carbonate could be proven to be more efficacious in the treatment of early acute glomerular nephritis than is magnesium sulphate over which it has definite advantages. It cannot, of course, replace injectable magnesium sulphate.

The few cases herein reported were so done for the purpose of stimulating interest in determining the true value of light magnesium carbonate.

*720 Farmington Avenue.*

---

CONGENITAL HYDROCEPHALUS IN INFANT. EPIDEMIC HEPATITIS IN MOTHER. (Acta Paediatrica, Stockholm, 40: 239, May 1951). The author reports on a male infant with severe hydrocephalus and microcephalus noted at birth. There was no evidence of syphilis or toxoplasmosis. The infant died when 8 weeks old. Post-mortem revealed an enormous hydrocephalus externus (1,760 cc. of fluid). The shape and size of the cerebellum, pons, and medulla oblongata were normal, whereas the cerebrum was a mass of white brain matter the size of a walnut. It is possible that the hydrocephalus was originally internal, since what was left of the cerebrum was a sac with signs of having been ruptured. The patient's mother had had epidemic hepatitis in the second and third months of pregnancy. The abnormal conditions in the child are taken to be the result of the acute infectious hepatitis in the mother. No similar case seems to have been published.—*Journal A.M.A.*

# PARATYPHOID (SALMONELLA) IN THE BAY REGION OF CALIFORNIA

J. C. GEIGER, M.D., DR.P.H.
Health Officer, Oakland, Calif.

During recent years numerous cases of gastro-intestinal disease, presumed to be infectious yet not acutely contagious, have appeared in various parts of the country. These cases, some possibly only accompanied by a cold and others with no symptoms of influenza, are frequently carelessly called "intestinal influenza."

We shall not resolve the group of gastro-intestinal diseases until we are in a position to use sound epidemiologic methods. The problem appears not to be one which can be settled by the laboratory, and clinicians cannot unaided separate the factors concerned. Epidemiologic work, like that of a detective, requires hours of painstaking search for facts, patience in seeking connecting links, an imperviousness to the red herrings that are continuously pulled across the path, and finally an accurate coordination of the whole.

Among the relatively mild or momentarily acute gastro-intestinal diseases, some of which are enduring and a few of which end fatally, segregation of the problems is difficult. One problem is certainly that of bacillary dysentery, caused by almost endless types of a group of bacteria called the shigellae. These can be identified often only in early laboratory specimens and then infrequently and for so short a period that these cases are usually never identified with certainty. A second problem is food poisoning. This disease, when it hits a few persons in a home or a small number of separated persons who partake of one product, is likely not to have either clinical or epidemiologic identification; even if such is made, it is likely to be without laboratory support. Food poisoning is a term which covers several kinds of bacterial upsets and also some troubles of nonbacterial origin which may occasionally complicate the story.

Intestinal parasites, protozoa or worms rather than bacteria, account for a few gastro-intestinal upsets. Even indigestion, allergy, fatigue, or nervousness can bring on symptoms which are likely to be called by any convenient name. The more we examine the situation, the more we wonder whether or not the term, intestinal influenza, should be conceded at all, yet the story is too ill-defined to rule out the existence of a specific infection, perhaps

due to a virus not recognized. We are quite sure of one disease which, infecting very young infants, has a high case fatality, is quite certainly specific, and is presumed to be due to a virus.

In short, there is a sporadic incidence of gastro-intestinal disease in which most of the cases are never identified and many are not reported.

In the "Bay Region," over the past ten months or thereabouts, there have been cases with diarrhea and varying symptoms of fever and distress from whom there have been recovered bacteria known to be one of the most common of what, because the disease resembled typhoid fever so much clinically, bacteriologically, and epidemiologically, was once called the paratyphoid group, the source of which was often traced to animals, frequently rodents.

It cannot be stated as a proved epidemiologic fact, but the story of cases in the "Bay Region" suggests that the most common of the salmonellae, as the members of the paratyphoid group are now called, *Salmonella typhimurium*, may be reaching persons from field mice or perhaps other rodents with results that are sometimes serious and which occasionally may spread through a family.

In San Francisco, between July and December 1951, there were 28 reported and hospitalized cases. Geographically, these were widely scattered the few exceptions appearing to represent related cases. Three sets of related cases indicate potential contagiousness, although this feature is not as pronounced as would be expected if we use typhoid fever as an analogy. One was a father and child, one a possible contact in an apartment house, and a third group centered around a Thanksgiving dinner, one of the cooks at which had been ill two weeks earlier. There were two deaths, both in older persons. About half the cases were under eight years of age, active children, some of whom were known to have played in vacant lots where rodents existed. Many of the premises were definitely clean, but there were noteworthy exceptions. However, grass fires, early rains, and cold drove numbers of field mice into homes.

In Oakland, nine cases were reported during approximately the same period, and there is no reason to believe that the situation is changing. Several of these also were related, apparently, but the situation appears to fit a postulate of a rodent-borne epidemic of low proportions, with widely scattered cases derived primarily from

rodents. Transmission from patients directly to other victims seems to depend on a chain of circumstances which precludes marked contagiousness.

Although these cases have been subjected to considerable study, both as to field investigation and laboratory examinations, the presence of both undiagnosed and unreported gastro-intestinal upsets, and the wide spread in both geography and time make it unwise to consider the situation as epidemioloically proved. Nevertheless, these cases were found and studied, and there were certainly others that were not. The existence of such cases was proved during the investigations by tracing points of contact and questioning members of family or neighboring groups. The reports must be regarded as only samples of what is actually occurring. The evidence is strong enough to suggest need for a renewal of efforts to control rodents, and a real effort to spot and consider epidemiologically all known cases of gastro-intestinal disease.

These cases all have causes, and many of them have epidemiologic significance. Unless they are detected and analyzed, we have no way of countering the normal risks as they ebb and flow.

The control of rodents is a civic responsibility, in which citizens have a part. There is also needed a consciousness of the problem on the part of physicians and citizens, so that the separate kinds of gastro-intestinal disease can be segregated and defined as well as possible, and so that the whole may not be smugly dismissed under the protean diagnosis, intestinal influenza. If that is an entity, which can be doubted but is at present neither proved nor disproved, then let it be a separate problem of the group and not a diagnosis that fictitiously pigeon-holes all undiagnosed upsets.

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DURATION OF LIFE IN CHRONIC LEUKEMIA. (Ugeskrift for Laeger, Copenhagen, 113:1304, Sept. 27, 1951). In most cases of leukemia in childhood the course is acute. Chronic myeloid leukemia is characterized by insidious onset, protracted course, marked enlargement of the spleen, no or slight peripheral adenitis, and pronounced increase of cell count in the peripheral blood. In only 2 of 71 cases of myeloid leukemia in children under 15 treated in Denmark from 1942 to 1949 was the course chronic.—*Journal A.M.A.*

# NATURE AND IMPLICATIONS OF THUMB-SUCKING*

## A Review

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Since the phenomenon of thumb-sucking may constitute a major factor in the behavior pattern of the child, one finds the act to be the subject of a great deal of discussion, it being considered in varying degrees of inclusiveness. Klackenberg[1] precisely terms it to be all habitual--sucking on thumb, other fingers, a lock of hair, a sleeve, a pillow, sheet, blanket, or toys—this sucking taking place while dozing off, or during sleep or at any time of the day when there arises need for solace. He excludes thumb-sucking which has been discontinued prior to one year of age, considering this to be an entirely normal phenomenon. Bakwin,[2] however, includes thumb-sucking at any age, stating that the habit usually becomes established during the early months of life, and finds that if the habit persists after one year of age, it is likely to run a long course, disappearing between two and five years of age, or persisting into adult life.

Associated movements frequently accompany thumb-sucking. The child may pull the ear, pat the head, twist or pull the hair, pull a blanket in front of the face, etc. The associated movement may persist after the thumb-sucking has ceased.

One may at this point wonder why so much attention should be paid to this act, but when one considers that sucking is probably the most important volitional motor activity of the newborn infant, and that the infant's survival depends upon his ability to suck, since it is through this function that he is able to ingest food, the investigator can readily see why much interest has centered around this act from the following viewpoints: 1. Its influence upon the physical development of the child. 2. The significance of thumb-sucking as a manifestation of the child's psychological development.

In an attempt to explain the influence and significance of this act, several hypotheses have been put forward to explain why children suck their fingers. Of these, the one by Levy[3] has received widest attention. On the basis of clinical investigations, as well

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as investigations in dogs, calves and chickens, he concluded that the primary factor causing thumb-sucking is inadequate sucking activity during feeding time. Corroborating this theory, Klackenberg, in judging from his series of 259 cases, concluded that one of the primary determinant factors in the development of the habit is the degree of satiation in infants of the sucking activity. In support of this, Klackenberg states that none of the at least four-year-old children who had used a dummy teat (more than ten per cent of the entire series) had ever been thumb-suckers.

Gesell and Ilg,[4] however, believe that inadequate sucking activity is of only minor importance. Gesell lays emphasis on the normal developmental trait which he calls hand-to-mouth reaction. When the maturation of the nervous system has advanced so far as the age of three or four months so that the infant can manipulate its hands and arms, there appears a normal tendency to raise them to the mouth and there liberate the sucking reflex. Thumb-sucking is thus, according to Gesell, an entirely normal phenomenon, a stage of development which should be passed by the second to third year of life. Sporadic relapses may occur on local irritation of the mouth, such as teething, or if, for some reason or other, the interest and attention of the child are directed from a normal and healthy extrospective activity to the child himself. Gesell thus does not attach much importance to the mode of feeding and the duration of the sucking phase of the feeding act. Bakwin's experience has been similar; he also finds that thumb-sucking is closely associated with sleep, and that many children suck the thumb only when falling asleep and during sleep.

As far as frequency is concerned, Klackenberg in his study reports that thumb-sucking and other comparable sucking occurred at the age level of one year in fifty per cent of the cases. At the age level of four years there was a frequency of 36 per cent. These figures included both the mild and more severe cases of thumb-sucking. Kaplan[5] feels, however, that although thumb-sucking is no longer prominent as an activity by the end of the third year of life, age cannot be used too rigidly as a standard because of variations in biopsychological development in relation to chronological experience. However, he thinks that one can reasonably state that when thumb-sucking persists or recurs at a time when the child is biologically capable of satisfying his basic needs in more compli-

cated ways, it may then be regarded as at least one indication of the child's difficulty or inability to accept or master the modes of obtaining gratification more in accordance with the biological maturity of his body.

Considering the physical and functional effects of thumb-sucking, it is found that it may displace the teeth, and it not infrequently causes callous formation on the thumb. Aside from these, the habit is not known to be harmful. It does not cause deformity of the palate, and air swallowing, oral infection and gastro-intestinal disturbances are no more frequent in thumb-sucking children, than in others. It is true that thumb-sucking and masturbation are both habitual manipulations of the body which are pleasurable, but there is no reason to believe that they are otherwise related. Moreover, there are no data to indicate that thumb-sucking children masturbate in later childhood more often than other children.

The psychological implications of thumb-sucking are of relatively greater significance. Kaplan notices that in many instances sucking movements will continue long after there has been an ample ingestion of food. From this, he deduces that sucking also occurs in response to a second impulse tending toward the satisfaction of another need, which can be described as a need for sensual gratification; and one may infer that the infant experiences a sensation of pleasure. The need for sensual gratification is intimately connected with, and tends to reinforce, the instinct for self-preservation, but continues to operate independently as well.

During the early period of the infant's life, it would seem as if the oral zone serves not only for the ingestion of food or for sensual gratifications, but also for the exploration of the nature of readily movable objects in his environment; thus he seems to explore his world by means of the sensations of the oral area. The infant soon learns that he can most easily obtain sensual gratification through sucking his thumb. To some extent this serves to make him independent of others in obtaining pleasure. Now the infant's satisfactions are intimately tied up with, and dependent upon another person, the mother. The earliest pattern of his relationship to the mother is established mainly around oral activities. The earliest satisfactions and frustrations experienced in relation to the mother center chiefly around this area of the body. We can thus begin to understand the significance of oral activity in the establishment of

important patterns of interpersonal relations, and are better able to appreciate the dual psychopathologic aspect of thumb-sucking:

1. In the first place, when needs of whatever nature are frustrated or inadequately met, all individuals tend to react with anxiety. In addition to the many maneuvers through which he attempts to overcome this uncomfortable feeling, the individual will also tend to adopt old, familiar, but outgrown patterns of behavior. Now sucking is one of the earliest patterns of behavior and perhaps the one most firmly established. Since the child has already learned during infancy that he can obtain through his own body some of the pleasures to be derived from sucking, he will frequently resume thumb-sucking during periods when he feels an increased amount of anxiety.

2. The other important psychopathologic aspect of thumb-sucking lies in its reflection of a deficiency in interpersonal relationships. The persistent thumb-sucker indicates in effect that he is unable to obtain necessary satisfactions by means of his accustomed ways of interreacting with other people, that to some extent he has at least for the time being, given up his attempts to obtain gratification through and with others; and to this extent he has withdrawn within himself.

When viewed from this background, persistent thumb-sucking can no longer be considered apart from the emotional state of the child, which necessarily demands consideration of his relationship with people significant to him. The focus of our attention must then shift from regarding it as a disease to seeing it as a symptom which indicates an emotional disturbance.

For the management of thumb-sucking, Bakwin advises that one should:

1. Discourage the use of bitter tasting substances and adhesive tape to the thumb.

2. Avoid censuring, nagging and shaming the child.

3. Review the handling of the baby with the mother to see that he is held properly during feeding, that he gets an adequate amount of manipulation (but not too much), that the mother knows the baby prefers to be held snugly, not loosely.

4. Not advise therapy during infancy or later on, when it is practiced only in relation to sleep, but that when the thumb-sucking is practiced during the waking hours after the first year of

life, it means that the child is over-fatigued, bored or unhappy. Therapy should then be directed toward correcting the total situation rather than the thumb-sucking. The child should have adequate rest and ample play outlets. An attempt should be made to correct undesirable prenatal attitudes.

Pursuing a similar approach, Klackenberg divides therapy into three classes: 1. Threats, nagging, admonitions. 2. Restrictive mechanical measures. 3. Attempts to cause distaste for the thumb by the application of bitter and nasty tasting mediums (pepper, etc.).

However, he does not think much of any of these measures, blaming them as a frequent cause of nervousness and restlessness. Instead, he suggests that there are usually a number of environmental factors that primarily should be corrected so that the child will be in less need of solace.

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PNEUMOTHORAX IN NEWBORN INFANTS. (Hoja Tisiológica, Montevideo, 11: 239, Sept. 1951). Pneumothorax may cause the death of a newborn infant without a diagnosis. It may occur in premature as well as in full term infants. Trauma, violent artificial respiration, and oxygen insufflation without control of oxygen pressure are the casual factors. Dyspnea and cyanosis are characteristic symptoms. Shock may follow rapidly. The diagnosis should be suspected in the presence of these symptoms associated with rales in the base and middle zone of the lung. The diagnosis is confirmed immediately by radioscopy and roentgen examination of the chest (front and profile views). Treatment of this emergency consists of immediate puncture of the thorax with slow withdrawal of air from the pleural space. A case responding favorably to this treatment is reported.—*Journal A.M.A.*

# CLINICAL REVIEW

*In order to encourage the writing of clinical articles by recent graduates or senior medical students, the* ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York, and Doctor Philip Moen Stimson, Cornell Medical School, New York. Other interested medical schools are cordially invited to submit student papers for consideration.*

## POLIOMYELITIS*

### A DISEASE PROCESS AND A COMMUNITY PROBLEM

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### THE DISEASE PROCESS

Poliomyelitis as a disease process is almost as complex as the field of new growths. In both there are many more or less well-established ideas as to etiology and pathogenesis, but no one specific idea that can be considered universal for the disease. The current theories as to the mechanisms involved are not difficult to enumerate, the task in polio, seems to be more that of placing these mechanisms in their proper sequence and assigning the proper importance to each. With the wealth of literature on the subject and the great amount of conflicting concepts, this is not an easy task and a paper of this type may unintentionally exhibit many of the prejudices of the authors on the subject.

Notwithstanding this drawback we have attempted to fuse the literature into a paper which will not only present some of the current ideas, but which will also place these ideas into a logical sequence for the better understanding of the disease process of poliomyelitis.

During the past few years, poliomyelitis has received increasingly more publicity in newspapers and magazines throughout the country. Rarely does a day go by during the summer months when one does not see articles in the newspapers and magazines advising parents how to protect their children from this scourge of childhood. Mothers are becoming increasingly more conscious of

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this threat to their children and more and more questions are being asked of the family physician on the subject of polio.

One may wonder at this interest in a disease which ranks low on the list of causes of death in this country and has a small incidence when compared to diseases, such as cancer or pneumonia. However, the fact remains that as yet polio. cannot be definitely controlled. It is the cause of almost one-fourth of the orthopedic deformities in children. An epidemic of polio. may easily run rampant through a community despite efforts by the local authorities. For this reason, polio. ranks as a potentially dangerous disease and will hold that rank until definite measures of prevention and control are established.

Since many problems of the disease process have been solved by modern research, it is not too much to expect that in the near future polio. will be entirely conquered and relegated to a comparatively benign place among the diseases of man.

*History of Poliomyelitis.* Poliomyelitis is not a new disease. It has occurred all over the world for some thousands of years but not in the epidemic form it has recently taken. A skeleton discovered in Egypt, which archaeologists assume to be of the period 3700 B.C., exhibits bone deformities which are indicative of the crippling effects of infantile paralysis. The designs on the Egyptian tombstones of the Prince Ruma from the 18th dynasty, 1580-1350 B.C. depict a withered limb with the typical appearance of poliomyelitic lameness.[1] Galen (131-200 A. D.) comments on attacks of paraplegia which he thought were brought on by the "cold winter winds succeeding a humid autumn."[2] Such scattered bits of evidence lend support to the notion that polio. is an ancient disease.

In later times several bits of historical literature strongly suggest that their subject matter dealt with what we would now think of as poliomyelitis. One of the most interesting of these is the account by Sir Walter Scott of his own illness, which was recounted to him by his parents. In it he states that as a child he awoke one morning with a fever that lasted for four days. On the fifth day he had lost the use of his right leg. In subsequent days, by slow stages, he was able to stand, walk and run and became again, with the exception of a partially lame leg, a healthy and high-spirited child.[3]

One of the earliest descriptions of poliomyelitis by a medical man was that published by Jacob Heine in 1840.[4] Heine was a German orthopedic surgeon who had seen many cases of deformed extremities in children. He perceived that the paralyses were the result of an acute disease of some sort which preceded the muscular weakness. However, the disease was not recognized in epidemic form until much later in the century.

In 1887, Oskar Medin,[5] a Swedish physician, observed the first sizeable epidemic of the disease. He was able to show that during the entire course of the malady the symptomatic phases of the disease, that is, the acute disease and the muscular weakness, belonged together. He further examined spinal cords at autopsy and was able to demonstrate the damage to nerve cells which controlled the muscles.

Paralleling these discoveries, similar observations were made in the United States. Colmer, in 1843,[6] described a teething paralysis of children under two years old in Louisiana. This was followed by Caverly's account in 1894 of an epidemic comprising 132 cases of poliomyelitis in Vermont.[7] He pointed out that the majority of these cases bordered on the bank of Otter Stream which flowed through the settlement, giving rise to the water-borne theory of poliomyelitis transmission. At the same time he presented further evidence that polio. is not always a paralyzing disease, and he spoke of the many cases which exhibited symptoms of the disease and yet did not develop paralysis.

Back again in Sweden, in the year 1909, began the first of a series of large epidemics of polio. in that country which were to continue right up to the present time. Ivar Wickman,[8] a pupil of Medin, established the epidemiological pattern of the disease from this first large epidemic of some 1,031 cases.

Wickman characterized polio. as an epidemic infectious disease primarily affecting children and spread by contact with clinical and subclinical cases or symptomless carriers. He was able to describe several secondary cases resulting from contact with infected individuals, for this epidemic involved several small villages and rural areas which could be extensively studied.

Once the infectious nature of the disease was established, many workers attempted to discover the transmitting agent. The first advance in this difficult quest was accomplished in Paris by Land-

steiner and Popper in 1908.[9] By injecting monkeys with spinal cord emulsions from fatal cases of polio. he succeeded in reproducing the disease on an experimental basis. This work was further collaborated by Flexner and Lewis of New York,[10] and Flexner further demonstrated that the disease could be caused by emulsions of spinal cords which were first filtered through earthen filters, thus establishing the viral etiology. With the etiology of the disease established, the door was opened to the intensive experimental studies as to its mechanisms.

*Viruses of Poliomyelitis.* The virus causing poliomyelitis is extremely small. Electronmicroscopic studies have shown that it ranges in size from 10-20 millimicra with a diameter of 12 millimicra.[11] The virus is hardy, for it has been preserved for years at temperatures from —20° C. to —70° C. Ether has little if any harmful effect on the virus, a characteristic which is especially useful in the laboratory for the preparation of contaminated material such as stools and sewage for inoculation into monkeys. Ph. ranges of 4-10, or 1% phenol, are unable to inactivate the virus.[12] On the other hand, it is easily destroyed by protein denaturing agents, such as heat, formalin, and potassium permanganate.[13] Unlike most of the viruses pathogenic for man, the virus of polio. will not satisfactorily grow in the embryonated egg.

Studies on the shape of the virus have been hampered by the inability to grow it in a concentrated preparation free from lipoid material. Preparations are usually made from feces or infected nervous tissue. The Lansing strain, recently examined by the electronmicroscope, appears to be a spherical or slightly asymmetrical particle.[11] Another member of the poliomyelitis group, Theiler's mouse encephalomyelitis, is 14x115 millimicra in size.[16] It is outstanding among animal and plant viruses in having a shape which is not a short rod like vaccinia virus, or a sphere like most animal viruses but one resembling the needle shape of many plant viruses, the tobacco mosaic being the classical example.

While there are an unknown number of types and strains of polio. virus, recent work by Morgan, Bodian and Howe[14] have done much toward furthering the classification of polio. virus by immunological methods. These workers, using cross immunization methods in monkeys, vaccinated animals with one strain causing immunity to that strain. These animals were then challenged with

another strain and the presence or absence of paralysis noted. In this manner fourteen strains of poliomyelitis virus selected from various parts of the country were placed into three distinct immunological types. More recently Kessel and Pait[15] further collaborated this work by challenging vaccinated and convalescent monkeys with heterologous strains, as well as carrying out neutralization tests with sera obtained from previously vaccinated monkeys. Constant results were obtained with all three methods, definitely confirming Morgan's work.

*Reservoir.* The reservoir for the poliomyelitis virus is generally believed to be man. Domestic animals, those of man's common environment, such as dogs, cats, chickens, horses, pigs and sheep, have all been demonstrated to be not susceptible to the human virus.[17] Recently, however, Hammond et al.[18] reported that 78 per cent of 55 cows, 63 per cent of 48 horses and 44 per cent of 112 domestic chickens were able to neutralize the Lansing strain of mouse adapted virus. Sabin,[19] in a series of experiments conducted on Okinawa, found that children developed antibodies against the virus at a very early age and that animals in this area, when tested with varying amounts of virus, were negative as compared to the results in the children. Hammond's results seem insignificant for the amount of virus was small and the amount of neutralization so slight that compared to human neutralization tests it is almost negligible.

Poliomyelitis virus has not been recovered from wild rats,[20] mush rats,[21] or in fish from water polluted with infected feces[22, 23] or from birds near polluted waters[24] or from cockroaches, ants, and mosquitoes near paralyzed patients.[25, 26] Sewage has been considered a possible reservoir for the virus. Many investigators have found virus in the sewage during epidemics[27] but not before the outbreak. Raw sewage has been found to have high activity, in some cases high enough to suggest that the virus was multiplying outside the body or that more people were excreting it.[28, 29]

The only other animal from which virus has been recovered in nature is from flies. During epidemics, flies caught both in and away from the polio. districts have been found to be carriers of the virus but without evidence of multiplication.[30, 33]

In the laboratory, however, Melnick and Penner[34] fed polio. virus in feces to blowflies and were able to demonstrate the virus

in the feces of these flies in some cases as long as three weeks after the feeding. This work suggests that flies are not only passive carriers, but that the virus may be able to multiply in these flies and to be excreted for as long as three weeks. A great deal of work must yet be done on this phase before one can reach definite conclusions.

The possibility that earthworms, frogs and plants or plant parasites may serve as a reservoir for the virus still remains to be explored, but at present man is still the only definitely known reservoir for the poliomyelitis virus.

*Source of Infection.* The most common potential sources of infection with the poliomyelitis virus are the urine, saliva, nasal secretions, pharyngeal secretions and feces. As yet, no virus has been found in urine, saliva or salivary glands,[35,36] hence one need only consider nasal and pharyngeal secretions and feces as possible sources.

It is unlikely that nasal secretions are often concerned because the seasons of polio. and upper respiratory infections do not coincide. Again, sneezing in polio. is very rare. In a controlled study on 22 paralytic patients,[36] pooled secretions collected from cotton plugs instilled deep into the nasal cavity for three days failed to yield virus even when collections were started three days after the onset of symptoms.

On the other hand, virus has been recovered from the pharynx by swabs, tissues or washings in 43 per cent of cases during the first few days of the disease, during the time that people in some outbreaks seem to be most infectious.[37,38] Virus has also been recovered from the throats of healthy carriers[39,40] and of patients from six days before the onset of the illness until 14 days after its onset.[41,42]

Virus has been found in the feces of nearly every patient when adequate technique is used. It has also been found in contacts with a frequency proportional to their intimacy.[43,44] Usually virus is found less concentrated in the feces of adults as compared to children and is not as continuously excreted.[45,46] However, virus obtained from the stools is able to infect monkeys by intranasal inoculation, and chimpanzees by mouth, nose, or stomach.[47,48]

It therefore seems fairly conclusive that the most probable sources of infection are from the discharges of the pharynx and

the feces. The comparative importance of these sources is yet to be evaluated, but it seems logical that pharyngeal secretions play a more important rôle in contact infections. Feces, on the other hand, may be considered the more important source in subclinical infections, non-contact infections and in the perpetuation of epidemics.

*Mode of Transmission.* The spread of poliomyelitis from one individual to another is possible in a variety of ways: indirectly, by an arthropod vector, through the mass contamination of food or fluid by feces, or flies, hand to mouth contact and lastly by droplet infection.

It seems that the first possibility can be quickly ruled out for poliomyelitis has no significant blood phase. Ward et al.[49] tested a series of 111 patients and were able to find virus in only one instance. Previous to this no one had reported polio. virus in the blood of patients with the disease. In addition, polio. is not associated with filth or poverty and it occurs in the winter when other insect-borne diseases of the nervous system, such as mosquito transmitted equine encephalitis, do not occur.

This seems to rule out the arthropod vector as a significant means for the transfer of the polio. virus.

There is a great deal of evidence to indicate that the mass contamination of fluid and food by feces or flies plays a major rôle in the transmission of poliomyelitis. As cited previously, many investigators have been able to isolate virus from feces, sewage and flies. Ward et al.[50] demonstrated that food exposed to flies in the home of polio. patients was also capable of producing the disease in chimpanzees.

Whether or not flies prove to be important, it seems certain that food is one of the important vehicles for the transfer of the disease.

During World War II, it was noted[51,52] in the middle east and India that the incidence of polio. among the enlisted men was less than a quarter of the incidence among officers. The enlisted men's kitchen was managed by British cooks or strictly supervised natives whereas the officers employed natives with little or no supervision and the kitchens were far dirtier.

It is known that mouse strains of virus may well survive pasteurization[53] and there are several epidemics on record where milk seems to have been the most probable means of transmission.[54,55]

Although polio. has never definitely been shown to be transmitted by drinking water or swimming pools, this possibility is borne out by the fact that the polio. virus in the presence of organic material is not susceptible to the usual amounts of chlorine used.[56] Again, several outbreaks of polio. have seemed to follow definite watercourses.[57]

These instances all favor food or fluid as a common vehicle for the transmission of the disease.

Person to person transmission of the virus is also a very possible means. If one watches children at play, one can see the many opportunities present for direct transfer of feces or pharyngeal secretions, not only from child to child but also to their adult associates.

Contact infection seemed to play the major rôle in outbreaks in the United States between 1941 and 1945 where the most important factor was close contact involving hugging, kissing, or playing together from a few days before to a few days after the onset of the illness.[58] In several instances there was a clear-cut history of only a few hours contact and in the case of very young children (1-3 years), the disease at times seems to be as contagious as mumps or measles.[58] One cannot overlook the presence of infection in all members of the same family at the time of its occurrence as evidence of either contact infection or common source infection.[59] Since it is common knowledge that virus can be demonstrated in the pharyngeal secretions of healthy people,[60] coughing and sneezing most certainly play a rôle in transmission.

It is evident, therefore, that the transmission of the virus varies both in and between epidemics. In some cases, personal contact seems all important, in others, fecal contamination of food is the major factor. Although flies and sewage are potential agents, direct proof is lacking. Flies may initiate the outbreak which may then be continued from person to person through the winter months by contact infection.

These several views on the transmission of the disease can be grouped together into three main categories as postulated by Sabin,[61] all of which regard the human as the reservoir. These views may be considered as the respiratory, alimentary, and the alimentary plus flies. According to the first, transmission occurs when the virus is breathed out of or expelled from the mouth or

nose of one person and is then breathed in by another. This hypothesis is based on the fact that (1) virus has been demonstrated in the nasopharynx and (2) that the infectious period of the disease corresponds more to the length of time that virus can be demonstrated in the pharynx, rather than to the much longer period of time that it is present in the feces. The alimentary hypothesis is based on the considerations that (1) the stool is the richest source of the virus, (2) the possibility of oral infection has been demonstrated experimentally, (3) lack of evidence to show definitely that under ordinary circumstances virus is expelled in droplets which can be breathed by someone else, (4) the fact that healthy carriers far outnumber the sick individuals and the infectious period of polio. cannot be defined as short by testing individuals who are sick and largely out of contact with other people a few days after the onset of the illness.

Neither of these views explain the fact that 90 per cent of polio. occurs during the summer and autumn. The alimentary plus flies theory regards the incidence of the disease during summer and autumn as a direct result of the wide dissemination of the virus by flies contaminated with human excrement. Small outbreaks may occur in the absence of flies and continue after the onset of cold weather due to the many carriers established by the initial dissemination.

From the public health point of view, it is of great importance to discover which of these views are closest to the truth. If the respiratory hypothesis is believed, one can get polio. without touching anything or anybody, or swallowing anything. Polio. would then follow measles and diphtheria as an epidemiological prototype.

On the other hand, in both the other points of view the epidemiological prototype is B. dysenteriae with its multiple immunological types and methods of spread.

*Portal of Entry and Route of Invasion.* In studying the pathogenesis of poliomyelitis one must be careful not to carry over experimental work done on monkeys to humans. In the earlier studies on polio, it had been shown by many investigators that in the cynamologous monkey the most natural route of invasion of the central nervous system was by the olfactory mucous membrane along the olfactory bulbs to the brain. Since man is so closely related to the experimental animal it was believed that the

portal of entry in man was also through the olfactory mucosa. However, Bodian and Howe,[62] and Sabin and Ward[63] by demonstrating the lack of lesions or presence of virus in the olfactory bulbs of humans have virtually ruled out that pathway to the central nervous system.

In man, the evidence points to the alimentary tract as the most logical site of invasion. It is here that secondary extension to the central nervous system occurs. Sabin,[64] in a systematic search of viruses in the tissues both nervous and non-nervous, found the virus localized in all levels of the alimentary tract. In the central nervous system virus was present only in certain areas: the motor cortex, the diencephalon, mesencephalon, medulla, pons and spinal cord. Such a distribution of virus is incompatible with a hematogenous spread but resembles a pattern of progression following the primary invasion of the spinal cord or medulla by way of regional nerves. The spread of virus from the oropharynx by nerve conduction is borne out by the evidence of polio. following the trauma of tonsillectomies.

Francis et al.[65] studied a family of eight in which five of the six children developed polio. following a tonsillectomy. The mother and father and one child, who did not have their tonsils removed, did not develop the disease. The stools of each member of the family as well as cousins who were visited before the onset showed the presence of virus. Since, as has been mentioned before, virus has been found in the tonsils and pharynx of healthy people,[66,67] the presence of the virus itself is not enough to cause manifestations of the disease, but following operation a direct extension along the cranial nerves (X and XI) to the brain occurs.

The centripetal spread of the virus takes place not only through the oral route but can also occur from the intestine. Burnett and Jackson[68] have recovered virus from autonomic nerves of the intestine after oral infection of cynamologous monkeys. In addition, virus has been recovered from sympathetic ganglia.[69] Thus one may say that after infection virus may progress to the central nervous system by either the sympathetic nerves of the intestine or by the autonomic nerve spread. As shown by experiments on monkeys,[70] the virus may reach the medulla or pons before the spinal cord is involved.

Centrifugal spread of the virus is also possible. Bodian and

Howe[71] found lesions in the trigeminal and spinal sensory ganglia due to centrifugal spread of the virus from the central nervous system. Weaver[72] found that after injection of the infraorbital nerve, virus could be recovered from the saliva, implying both centripetal spread to the central nervous system and then centrifugal spread to the periphery.

In the utilization of regional nerves for its invasion of the central nervous system, the larger caliber fibers offer best pathways for the virus. Bodian and Howe[73] have shown that after immersion of the hypoglossal nerve in a solution of virus for 10 minutes, bulbar paralysis occurred in four days, whereas similar immersion of the sympathetic trunk produced no paralysis in the same length of time. The virus actually utilizes the axis cylinder for its progression since in experiments in which the nerve has been frozen, no paralysis occurred even though physical contact was present. Living nerve fibers are therefore essential for the transmission of the virus.

In man the virus is disseminated through several preferential pathways. From the spinal cord it ascends by way of the corticospinal tracts to the motor cortex. Along the reticulospinal tracts it reaches the midbrain where, through many collaterals, it becomes widely disseminated. The cranial nerves leading from the oral cavity, pharynx and gastro-intestinal tract are also among the most likely routes along which the virus progresses to the central nervous system.[73]

Once in the central nervous system, the virus has certain affinity for some nerve cells, while others are refractory to invasion. The anterior horn cells are particularly susceptible, destruction of them leading to flaccid paralysis of skeletal muscles. In the higher centers, characteristic lesions can be found in the vestibular nuclei, reticular formation of the pons, motor cortex, globus pallidus, substantia nigra, red nucleus, and tegmentum of the midbrain. The lateral geniculate bodies, inferior olive and cerebellar cortex are entirely refractory to the invasion.[74] The wide distribution of the lesions explains the diversified symptoms that may be encountered in polio.

*Pathology.* The neuronal destruction in poliomyelitis is only a small part of the cellular reactions that occur in response to the viral invasion. Following infection of the nerve cell, an inflammatory reaction occurs with the mobilization of great numbers

of neuroglial and inflammatory cells. One may well wonder if the degeneration of the nerve cells is primary or secondary to the inflammatory process. This is evidently a primary factor in the understanding of the pathogenesis of polio. as a nervous system disease, for the virus is neurotropic or rather neuronotropic in that it attacks the nerve cell alone. Bodian and Howe,[73] in order to clarify this problem, inoculated the thalmus of rhesus monkeys with a virus suspension after left hemidecortication. In this manner the cells of the thalmus on one side had undergone degeneration and were nonfunctional upon inoculation. Microscopic examination disclosed a striking difference in the distribution of the lesions. There was a great preponderance of perivascular infiltrative lesions in the undegenerated nuclei of the right side whereas the nuclei of the other side showed few lesions.

It would appear therefore that the virus of polio. has a selective affinity for the nerve cell itself. As a result of this affinity, a chain of inflammatory phenomena occur involving both mesodermal and neuroglial elements, characteristic of the disease.

A reaction of this type would be most conducive to the destructiveness of the disease because before the mobilization of the phagocytes can occur the virus must exert its destructive action on the nerve cell producing possibly a chemotaxic substance which initiates the inflammatory response. This would differentiate the virus from pathogenic bacteria which excite a cellular response by their mere presence in the tissues.

The lesions of polio. cannot always be directly correlated with the clinical manifestations. In occasional cases the virus may wipe out all the motor neurones leaving the patient little or no voluntary musculature, but it is more common for the disease to be spotty, with certain muscle groups or even entire extremities completely spared. A constant finding in experimental polio. and in fatal cases is that the lesions are more widespread than would be predicted on the basis of clinical findings.[75] This is due to the fact that destruction of neurones may occur without producing disability if it is scattered in the anterior horn and not concentrated in a neurone pool which enervates a functionally related group of muscles.

Again, in the higher centers, lesions may be present in functional areas without symptoms of injury of such areas. Such

lesions including neuronal destruction and inflammation have been found in individuals who have never exhibited any symptoms.[75]

It seems that injury of the higher centers must reach a certain threshold of severity, this threshold varying with the area involved, before a clinical effect is observed. It is generally agreed that all cases of poliomyelitis are encephalitic, that is, lesions are found in the higher centers when only paralysis of the limbs are present. The anterior horn cells have the lowest threshold to destruction by the virus.

The occurrence of paralysis and recovery is dependent on the severity of injury to the anterior horn cells. In the course of the disease in which these cells are affected, there occurs first a reduction in size and staining capacity of the Nissl substance. This is characteristic of the so-called preparalytic period of polio. and if the process is arrested here, no paralysis results—an abortive poliomyelitis. If the process continues with severe chromatolysis and nuclear changes, necrosis occurs. It is noted that the anterior roots degenerate only when the cell body reaches the necrotic stage.

In the nonparalytic cases as well as the paralytic, Bodian[76] has shown that the extensive distribution of the lesions is similar. The difference in types is not in the number of nerve cells invaded but rather in the severity of the injury to the invaded nerve cells. Cells which are chromatolyzed by virus are either quickly destroyed in the first few days of the disease or undergo recuperative changes leading to complete recovery.

*Summary.* The importance of poliomyelitis lies in the paralysis and deaths it causes and is emphasized by the fact that recently in Minnesota 31 persons per 10,000 under the age of 21 were found permanently paralyzed.[77] The occurrence of nonparalytic polio. has been noted since 1896, its incidence having been estimated as from 3-100 times that of paralysis.

The main reservoir for the polio. virus is considered to be man. In the majority of cases the virus gains entrance by mouth either by food, fingers or respiratory contact. Although animals have been experimentally infected through skin and eye,[78] this cannot be generally considered an important mechanism in man.

It seems that most people exposed to the disease become infected, that in the greater majority of cases the virus spreads to the central nervous system, but that most of the cases do not become paralyzed.

Once the virus gains entrance into the body it localizes in various levels of the alimentary tract. From there the virus may leave the body through the feces, it may remain in the intestine with no nerve invasion, or it may invade the spinal cord and medulla by way of regional nerves.

If the virus remains in the alimentary tract without invasion of the central nervous system, few if any of the symptoms of the illness occur. It may persist in the so-called carrier stage until some trauma, such as dental operations or tonsillectomies make the conditions right for the invasion of the nervous system.

Once the central nervous system is reached, spread of the virus may occur either up to the brain from the cord or in the opposite direction. Depending on the severity of the injury to the nerve cells and the distribution of these injuries, a severe paralysis may ensue. If the destruction of the neurones is halted before irreversible changes occur, only a mild weakness or transitory paralysis will develop. Finally, if an insignificant amount of virus is present there may be no signs of involvement but rather an equilibrium between virus and host. This equilibrium may be upset by exercise, debilitation, pregnancy, secondary infection or other unknown factors resulting in either of the previous mentioned courses.

Because of the difference in virulence of the strains, as well as the amount of natural immunity present in individuals, it is difficult to give an exact estimate of the incidence of the forms of polio. However, on the basis of the above possibilities, about six weeks after exposure to the disease, .2 per cent of the intimate contacts would be dead, 2.8 per cent paralyzed, and a few paralyzed but able to recover.[62]

Of those not paralyzed, a few will later become paralyzed, while a small percentage will have a subclinical loss of motor neurones. The greater majority will have a central nervous system infection as shown by spinal fluid changes and meningeal signs; a smaller group will have only an alimentary infection with transient presence of virus in the feces. Finally, there will be some with no infection at all because of no symptoms, no signs, and no virus.[62]

*(Conclusion of paper, also References, will be published in August issue.)*

# PEDIATRICS AT THE TURN OF THE CENTURY

***From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.***

## THE ETIOLOGY OF ENDOCARDITIS IN CHILDHOOD*

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*Occurrence.* Endocarditis may occur in fetal life and may then be a cause of cardiac malformation. In fetal endocarditis usually the right side of the heart is affected, from which circumstance it follows that in infancy the presence of right sided cardiac disease is not uncommon. The pathological changes in these cases include thickening, shortening, and agglutination of the valves. Thus, two of the semilunar valves may be adherent or a set of valves may be firmly fused, leaving only an aperture in the center.

*Congenital Endocarditis.* While fetal endocarditis may be a primary cause of congenital defects these, once established, are certainly a condition predisposing to attacks of endocarditis in childhood (endocarditis recurrens). Acute endocarditis may originate in early infancy, but this is rare. Holt says that in the autopsies of over one thousand infants less than three years of age not a single case of acute endocarditis was found. Likewise Northrup and O'Dwyer discovered in 2,000 autopsies at the New York Foundling Asylum only 1 case which presented acute inflammatory lesions.

*Acquired Endocarditis.* After the fifth year of age endocarditis is not uncommon, and in later childhood it is of frequent occurrence.

*Rheumatism* is, as in adults, the disease associated with the greatest number of cases, but in childhood endocarditis is present in a far greater proportion of rheumatic cases than in adults. This is due, most probably, to the more delicate organization of the endocardium at this tender age, with a corresponding dimin-

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ished power of resistance. The severity of the general rheumatic manifestations does not afford an index to the liability of cardiac implication. In fact trivial articular disturbances are often attended by decided pathological changes in the endocardium. It is not necessary that the articular symptoms should precede the endocarditis. Endocarditis may be the first, and possibly the only, manifestation of the disease. Rheumatism furnishes more cases of endocarditis than all other conditions combined; and the proneness of acute articular rheumatism to make its appearance in late childhood explains the comparative frequency of endocarditis at that time of life. Endocardlitis is not to be regarded as a result, but as a manifestation of rheumatism; and is probably caused in these cases by a micro-organism—as yet unidentified—which, having a predilection for serous surfaces, attacks the endocardium just as it does the synovial membranes of the joints.

*Chorea,* which is closely related to rheumatism, if not indeed the same disease, is, especially in children, a not infrequent concomitant of endocarditis. Here again the process may be rather a manifestation than a result of the disease. Nor should the occurrence of endocarditis with tonsillitis be left unnoted in this connection.

*Scarlet Fever, Measles, Diphtheria.* All of the acute infectious diseases may be starting points for endocarditis. In these instances, however, as in pneumonia and similar diseases, the process is readically different from that existing in rheumatism and chorea. Here two factors operate; first, the disease by its increased demands upon the heart as well as by the vitiating action of the fever and the presence in the circulation of noxious substances impairs the resistant powers of the heart; and secondly, the weakened endocardium may then be successfully attacked by the specific bacilli of the special disease or by other agencies opportunely chancing to be present.

*Malignant Endocarditis* is exceptional in infancy. Harris' case in a child four years old was long the youngest case recorded. Malignant endocarditis may, however, occur in early infancy, as, for example, in my case of pyocyaneus endocarditis, where the patient was but two and a half months of age. In this case the pycyaneus bacillus, a germ ordinarily nonpathogenic or at most mildly virulent to the human species, not only invaded the circulation, causing a septicemia, but the circumstances being favor-

able, even succeeded in securing a foothold in the endocardium and in causing a malignant endocarditis. The favorable circumstances referred to were to be found in the debilitated condition of the infant (which was afflicted with hereditary syphilis). Two days before death, the heart sounds until that time having been normal, a loud, rough systolic murmur became audible. The temperature assumed an intermittent character; diarrhea ensued; and hemorrhagic bullæ suddenly appeared on the toes. In blood taken antemortem rod-shaped bacilli, which subsequently proved to be pyocyaneus bacilli, were discernible. From blood obtained from the heart one and one-half hours after death pure cultures of pyocyaneus bacilli were propagated. With the bacilli thus obtained a pyocyaneus endocarditis in a rabbit was caused experimentally (in the usual manner, viz., by first puncturing the aortic valves with a probe introduced through the carotid and, subsequently, injecting the bacilli into the circulation). In the pinhead sized verrucosities found on the infant's mitral valve I demonstrated colonies of pyocyaneus bacilli.

At this point it may not be out of place to touch upon the relation existing between the several pathological and clinical varieties of endocarditis. The lesions described as verrucosities, ulcerations, perforation, thickening, contraction and so on are not separate and distinct pathological processes but different stages or terminations of the same process.

Weigert, Fleischhauer, Eberth and others have proven that the primary effect of bacterial activity is a necrobiosis. This, then, in the endocardium may result in ulcerative endocarditis; but if the reparative forces are given opportunity to act in these cases, cell proliferation follows with a resulting cicatrix and shortening or thickening and the formation of verrucosities. Again verrucosities may be the primary pathological alteration and by degeneration they may give place to ulceration. Or, if the ulceration continues to extend, perforation may result. The proliferative process may begin within a few hours and usually does begin within twenty-four hours.

Malignant endocarditis differs from the great majority of cases of simple endocarditis only in the gravity of the constitutional symptoms. Thus rheumatism and chorea may be etiological factors in the causation of both varieties; while viewed from the pathological standpoint a severe verrucose endocarditis due, for

example, to the pneumococcus may be just as malignant as is ulcertative endocarditis. It may in fact be a stage precedent to the ulcerative process.

Endocarditis may be acute or chronic. In some instances it may be primary, but in most cases is secondary to an infectious process.

*Primary Endocarditis.* Luschka was the first to describe certain hemorrhagic tumors at the margins of the heart valves in new-born infants. More particularly he described these bullæ as occurring on the mitral valves. Parrot made a thorough investigation of this subject. He observed that hemorrhagic tumors from the size of a pinhead to that of a pea were not infrequently to be found at the margins of the valvular segments. These extravasations, which he attributed to the rupture of intravalvular blood vessels, are covered by the endothelial membrane. They occur more frequently at the mouths of the great vessels. Tracing their subsequent course Parrot observed that they might proceed in either of two ways. (1) By a process of absorption they sometimes disappeared with consequent thickening and induration. (2) Their contents became replaced by a hard mass. In the latter group of cases the primary stage of transition consisted in a proliferation of connective tissue cells at the margin of the tumors, this stage being succeeded by organization of the tumor mass. It is evident that this process, resulting in shortening and thickening of the valve segments and in the production of verrucose lesions of endocarditis, is purely reactionary in nature and that it is uncomplicated by foreign agencies such as bacteria. It is a factor and was recognized as such by Henoch, although latterly habitually disregarded—in the etiology of endocarditis.

*Bacterial Agencies.* On the other hand, the activity of bacteria in the causation of endocarditis is established beyond question.

Vieussen's discovery, in the latter half of the seventeenth century, of the anatomy of the heart gave the required impetus to the study of this organ. Immediately diagnostic means were sought, with the result that palpation, percussion and auscultation were quickly introduced. Albertini, Corvisart, Laennec, Skoda and many others studied cardiac symptomatology and pathology. Rheumatism and scarlet fever were recognized as etiological factors by Kreisig and Bouillard.

In 1855-1856 Rokitansky and Virchow published the first reports

of microscopical observations in endocardial lesions. But to Winge and Heiberg is due the credit of giving the first definite information. Winge's case (1869) was a malignant endocarditis following an abscess of the foot; Heiberg's (1872), a malignant endocarditis secondary to puerperal septicemia. In both cases the microscope revealed entities which in the light of our present knowledge were probably micrococci, but which the discoverers believed were leptothrix chains. In 1873 Eberth designated 2 cases as diphtheritic. Various investigators in the next few years reported cases of bacterial endocarditis. Klebs in 1878 published 27 cases in which he had found bacteria. In 1881 Litten attributed all cases of endocarditis to bacteria. Streptococci and staphylococci were positively identified in endocarditic lesions by Philippaux in 1885. Since that time the variety of bacteria recognized has constantly increased—including the pneumococcus, gonococcus, tubercle bacillus, and pyocyaneus bacillus..

*Experimental Endocarditis.* Winge and Heiberg introduced particles of the embolic masses obtained from their cases into the bodies of rabbits and failed to cause endocarditis (no vulnerable point existing in the endocardium). By mechanically wounding the cardiac valves and then introducing bacilli into the circulation, Rosenbach, Wyssokowitsch and Orth, Pruden, Michaelis and others caused endocarditis with widely different bacilli. My own series of experiments embrace positive results with the streptococcus, staphylococcus, pneumococcus (all previously performed by others), and tubercle bacillus, typhoid bacillus, bacillus pyocyaneus (original cases).

Bacterial endocarditis occurs in childhood as in adult life. In fact, as I have previously remarked, the delicate endocardial tissues of childhood offer less resistance to attack than do the mature structures of adult life, and are consequently an easier prey for bacteria. The extensive researches in this field indicate that many bacilli possess the power of successfully attacking the endocardium—probably all varieties which for any cause are pathogenic for the individual and which come into contact with the endocardium under favorable auspices; that is when a locus minoris resistentiæ is present.

In a previous paper, "The Etiology of Endocarditis with Especial Reference to Bacterial Agencies," I have reached certain

general conclusions as to the various causes of endocarditis and have presented therein the following scheme of classification: (1) Congenital and infantile endocarditis (defective development, simple reparative endocarditis—and unknown causes). (2) Endocarditis due to known bacterial agencies (streptococcus, staphylococcus, tubercle bacillus, pyocyaneus bacillus, etc.) (3) Endocarditis associated with definite diseases presumably of a bacterial nature; but of which the bacterial agents are still unknown (rheumatism, chorea, syphilis, the exanthemata, etc.). (4) Endocarditis due to mechanical or chemical insults (blows, strains, excretory products, alcohol, atheroma, etc.).

While examples of all of these classes may occur in infancy and childhood, the relative frequency of their occurrence presents decided variations from that existing in adults, and the different conditions prevailing likewise exert a decided influence.

In childhood the endocardium, as all of the tissues, has less power of resistance than in adult life and accordingly succumbs to attack more readily. Again, congenital defects sometimes exist which at once furnish a weak spot vulnerable to intercurrent factors. Children are the chief sufferers from the exanthemata and endocarditis secondary to this group of diseases is practically exclusively limited in its inception to the epoch of childhood. About puberty great demands are made upon the heart, and here again the endocardium may be affected. There is in infancy a not insignificant number of cases of congenital endocarditis of a severe grade which never attain adult life. In fact of the severe cases of congenital endocarditis, others than those implicating the pulmonic orifice rarely exceed the age of twelve. The predisposition to occurrence in certain conditions—for example, with rheumatism—is vastly greater in childhood than in adult life. Malignant endocarditis is exceptional in infancy and rare in later childhood; when it does occur it is most frequently associated with rheumatism. From the atheromatous ravages of advanced age alone the child completely escapes. But the effect which depraved systemic conditions in the mother—alcoholism, metallic poisoning of the blood, etc.—may exert on the endocardial structures of the fetus can at present only be conjectured. That bacterial endocarditis may occur in the embryo I see no reason to deny.

# DEPARTMENT OF ABSTRACTS

RATNER, B.; UNTRACHT, S. AND HERTZMARK, F.: ALLERGY TO VIRAL AND RICKETTSIAL VACCINES. III. INFLUENCE OF REPEATED INOCULATIONS ON THE ACQUISITION OF EGG ALLERGY. (New England Journal of Medicine, 246: 533, April 3, 1952).

In only 5 of 319 nonallergic children, subjected to many repeated inoculations of influenza and mumps-virus vaccines, did a dermal sensitivity to egg white develop. In no case was clinical sensitivity induced. There was no enhancement of clinical sensitivity in egg-sensitive children repeatedly inoculated with crude and refined influenza-virus vaccines. Egg sensitivity was not acquired after repeated therapeutic injections in 120 allergic children sensitive to substances other than egg. Within the limits of our data, the hazard of sensitization from repeated reinjections of viral and rickettsial vaccines seems negligible. This, we believe, results from the resistance acquired from the habitual ingestion of eggs and egg-containing foods. AUTHORS' SUMMARY.

SIEGEL, A. C., NICKERSON, G. H. AND COOK, C. D.: THE RECTAL ADMINISTRATION OF AUREOMYCIN IN CHILDREN. (New England Journal of Medicine, 246: 447, March 20, 1952).

Twenty children, ranging in age from 4 years to 12 years, were given aureomycin in doses of 20-80 mg./Kg./dose. In spite of the large doses the serum levels of the blood were variable and most showed inadequate serum levels of aureomycin. About three-fourths of the subjects had tenesmus and pain and about a fourth expelled the aureomycin within one hour. The authors conclude that, because of the poor absorption, wide variance in the serum levels, unpleasant and toxic side-effects and the large dose required and that despite the occasional achievement of low therapeutic levels rectal administration of aureomycin is an unsatisfactory method of treatment. MICHAEL A. BRESCIA, M.D.

RUSSELL, L. B. AND RUSSELL, W. L.: RADIATION HAZARDS TO THE EMBRYO AND FETUS. (Radiology, 58: 369, March 1952).

It has been shown that several mammals are highly susceptible to the induction of malformations by radiation. There are so-called critical periods in the developmental stage and this critical

period in man is estimated to be the second to the sixth week of gestation. In view of their observations, the authors recommend: (1) Whenever possible, irradiation involving the uterus in women of childbearing age should be restricted to the two weeks following the last menstrual period, to preclude the possibility of fertilization having taken place. This applies particularly to diagnostic irradiation, even if the doses involved are less than 25 r. (2) The present practice of avoiding irradiation during a known pregnancy, i.e., during later stages, should not be relaxed. (3) Whenever possible in non-pelvic irradiation, the conceptus should be shielded.

MICHAEL A. BRESCIA, M.D.

STEFANINI, M.; SANTIAGO, E. P.; CHATTERJEA, J. B.; DAMESHEK, W. AND SALOMON, L.: CORTICOTROPIN (ACTH) and CORTISONE IN IDIOPATHIC THROMBOCYTOPENIC PURPURA. (Journal American Medical Association, 149:647, June 14, 1952).

Eleven patients with "acute" and "chronic" idiopathic thrombocytopenic purpura were treated with ACTH or cortisone, and systematic studies were conducted in eight. Although no permanent remission of the disease was obtained in any of the patients studied, these hormones (a) reduced, in most cases, the spontaneous bleeding manifestations, (b) improved the capillary fragility, and (c) shortened the bleeding time and prolonged the phase of initial vasoconstriction that followed the incision of the skin. It appears that ACTH and cortisone may be usefully employed in patients with idiopathic thrombocytopenic purpura: (a) to control severe bleeding, especially in those with the "acute" type of the disease, when an expectant attitude may be rewarded by the occurrence of spontaneous remission, (b) prior to splenectomy, when this procedure is considered necessary, to reduce the loss of blood at operation, and (c) when splenectomy has been unsuccessful and severe spontaneous bleeding manifestations continue.

AUTHORS' SUMMARY

BROWN, M. R.: THE MECHANISM INVOLVED IN POLYNEURITIS AS EXEMPLIFIED BY POSTDIPHTHERITIC POLYNEURITIS. (Annals of Internal Medicine, 36:786, March 1952).

Postdiphtheritic polyneuritis in 53 patients has been remarkably consistent in its course. There is a weakness of the palate about

10 days following the acute infection. About 4 to 5 weeks later the patient experiences numbness and tingling of the arms and legs, and the deep reflexes cannot be obtained. Seven to 8 days after these symptoms, peripheral sensory loss and weakness of the extremities and even of the trunk muscles follow. At the same time, other cranial nerve palsies develop in about two-thirds of these patients. The nerves to the eye muscles are particularly susceptible to the diphtheria toxin. The spinal fluid exhibits the Guillain-Barré syndrome of an increase in protein without an increase in cell count more consistently than does the disease described by Guillain and Barré themselves. The mortality rate was 9.4 per cent. All the fatalities occurred in patients who had received antitoxin either late in the disease or not at all. Paralysis was also seen when the correct diagnosis had been delayed or missed. In this series, postdiphtheritic polyneuritis was not decreasing in frequency, and in recent years the indiscriminate use of antibiotics is increasing the number of delayed diagnoses.

AUTHOR'S SUMMARY.

CAPPS, R. B. AND STOKES, J.: EPIDEMIOLOGY OF INFECTIOUS HEPATITIS AND PROBLEMS OF PREVENTION AND CONTROL. (Journal American Medical Association, 149: 557, June 7, 1952).

An epidemic of infectious (epidemic) hepatitis among student nurses resulting from an epidemic focus among children patients under three years is reported. It is clearly shown that transmission from child to nurse occurred only by the fecal-oral route and from child to child probably in the same manner. The disease in the age group under three years is generally without jaundice and may persist for 6 to 8 months. Experimental evidence of a fecal carrier state 5 to 15 months duration was obtained through the use of adult volunteers. Prevention and control of epidemics in the light of available knowledge are discussed.

AUTHORS' SUMMARY

DICKIE, A. W.: TUBERCULOSIS IN FAMILIES WITH BCG—VACCINATED AND NON-VACCINATED CHILDREN. (British Medical Journal, 4760:682, March 28, 1952).

A description is given of 16 families in which some children were vaccinated with BCG and others not, and in which all had

been exposed to a known source of infection. Reasons are put forward for considering the two groups comparable. Among the 22 children in the non-vaccinated group, 21 developed primary tuberculosis. In the BCG vaccinated group, two at most showed evidence of superinfection. The protective value of BCG vaccination is striking, both when the source of infection is within the family (with the maximum risk for each child) and when it is outside. The difference in the incidence of tuberculosis in the vaccinated and non-vaccinated children admitted to Nortull's Hospital is recorded and a brief description of the fall in the number of cases of primary tuberculosis from 1945 on is given. The absence of tuberculous meningitis and miliary tuberculosis in the vaccinated group is noted.

AUTHOR'S SUMMARY.

PAUL, O., AND HARRISON, C. J.: WOLFF-PARKINSON-WHITE SYNDROME IN AN INFANT. (Journal American Medical Association, 149:363, May 24, 1952).

A normal 6 pound male infant was delivered uneventfully at term. At the five week check up examination an extremely rapid regular heart rate, estimated at 300 beats per minute, was found. Re-examination three days later revealed a rate of 120 beats per minute. His color appeared normal until he cried, when cyanosis of the forehead and circumoral region was seen. An electrocardiogram revealed a supraventricular tachycardia with a rate of 300. The infant developed congestive heart failure which was controlled by digifolin and other supportive measures. The diagnosis of W-P-W syndrome was made on the basis of the supraventricular tachycardia, the short P-R interval with the QRS interval actually longer than the P-R interval, and the typical slurring of the ascending limb of the R waves.

MICHAEL A. BRESCIA, M.D.

THE HAZARDOUS FIRST MONTH OF LIFE. (Statistical Bulletin, Metropolitan Life Insurance Company, 33:1, Feb. 1952).

About 78,000 babies under one month of age die in a year in the U. S. A. One-third of all deaths in the first year of life happen on the day of birth; three-fifths of the infant deaths occur in the first week. Premature birth is the largest single factor in neonatal mortality, accounting for about one-half of all the deaths

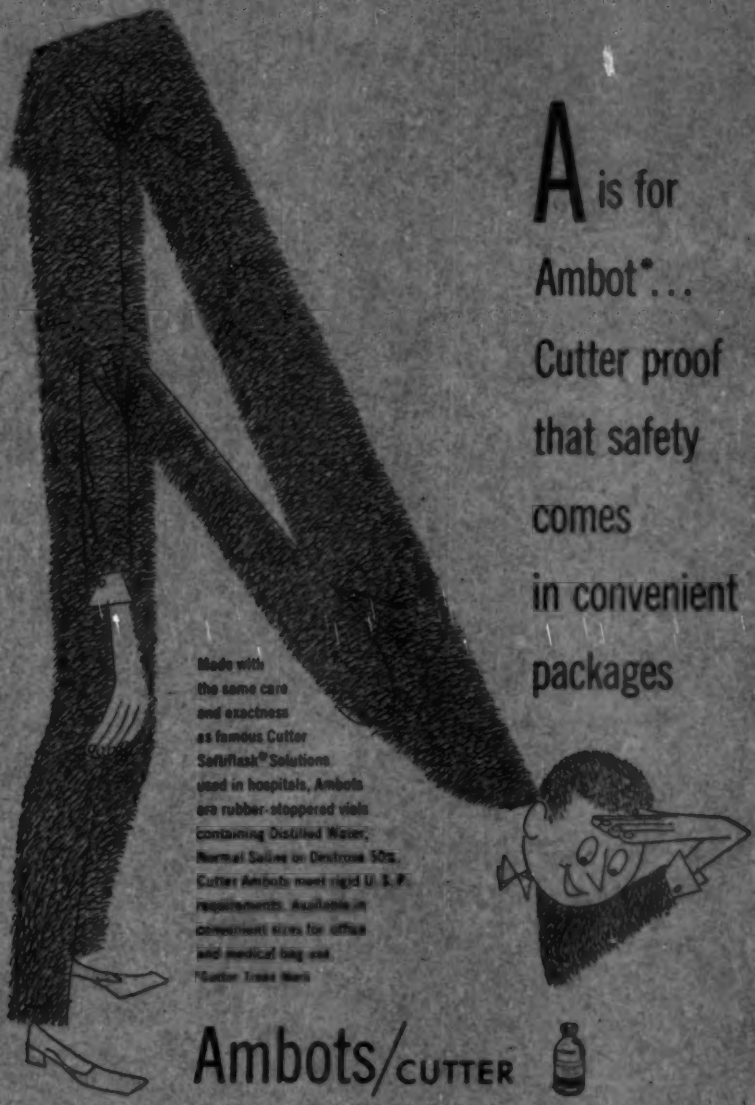
under one month of age. A majority of the other deaths are reported as due to injury at birth or congenital malformation. Death rates are extremely high among the lightest weight groups. Thus, of the infants who weigh 1,000 grams or less at birth, 95 per cent die in the first month of life. The mortality decreases progressively, however, with the more mature development of the fetus. Of infants in the 2,000 to 2,500 gram class at birth, the neonatal mortality was 6 per cent. Full term infants, those over 2,500 grams, had a death rate of less than 1 per cent.

MICHAEL A. BRESCIA, M.D.

RUBIN, S. AND STRATEMEIER, E. H.: INTRATHORACIC MENINGOCELE. A CASE REPORT. (Radiology, 58:552, April 1952).

A case of intrathoracic meningocele is reported, believed to be the fourteenth in the literature. Roentgen findings common to most of the cases are kyphoscoliosis, a posterior mediastinal mass projecting laterally through the intervertebral foramen, with enlargement of the foramen and absorption of the adjacent posterior ribs. In several cases, including the authors', there was evidence of multiple vertebral anomalies, such as bifid vertebrae. Nine of the 14 cases were associated with Recklinghausen's disease. The association with the latter condition of a posterior or paravertebral mass, enlarging the foramen and causing absorption of the adjacent ribs, should suggest meningocele rather than neurofibroma. Relative freedom from neurologic signs or symptoms is further evidence in favor of a meningocele. If the mass remains stationary in size, treatment should be conservative. If there is evidence of growth, surgical excision with closure of the dural sac is indicated.

AUTHORS' SUMMARY.



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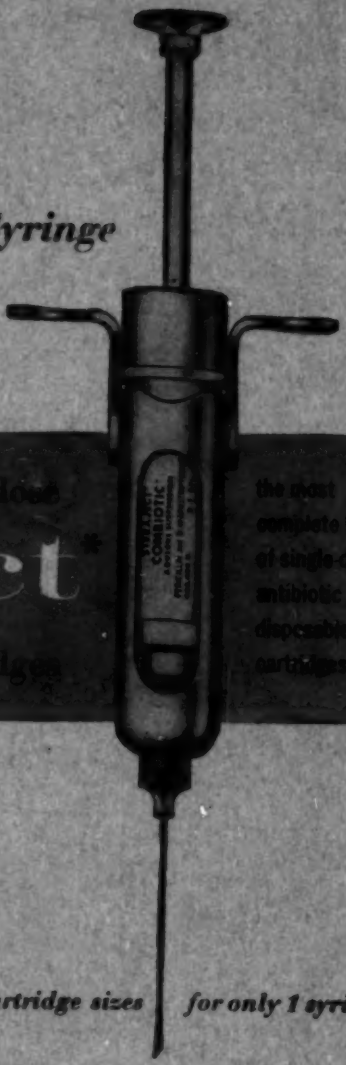
**holds 2 cartridge sizes**



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**Steraject Combiotic* Aqueous Suspension (400,000 units Penicillin G Procaine Crystalline, 0.5 Gm. Dihydrostreptomycin)**

**Steraject Dihydrostreptomycin Sulfate Solution (1 gram)**

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*2 cartridge sizes for only 1 syringe!*

two cartridge sizes permit *full standard* antibiotic dosage

cartridges individually labeled

ready for immediate use

no reconstitution

*for full details, ask your Pfizer Professional Service Representative*

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